First-in-Man Implantation of Left Ventricular Partitioning Device in a Patient With Chronic Heart Failure: Twelve-Month Follow-up

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ABSTRACT

Background: The ventricular partitioning device (VPD) (Cardiokinetix Inc., Redwood City, Calif) is a novel device that is deployed percutaneously in the left ventricle in patients with anteroapical regional wall motion abnormalities after a myocardial infarction (MI) to partition the ventricle and segregate the dysfunctional region. In this case report we present the first implantation of the VPD in a human, with a 12-month efficacy and safety follow-up.

Methods and Results: A 48-year-old man had an anterior MI in 2004. A coronary angiogram showed an occlusion of the proximal segment of the left anterior descending artery with no stenosis on other major epicardial vessels. Echocardiography revealed a dilated left ventricle (62 mm) with anteroapical wall motion abnormalities, no apical thrombus, a calculated ejection fraction of 26.8% (by Simpson biplane formula), and an end-systolic volume index (ESVi) of 76.8 mL/m². The VPD implant was delivered percutaneously from the femoral artery by the standard techniques for left-sided heart catheterization. The postimplantation course was uneventful. Echocardiography on discharge showed the VPD implanted at the apex, with a left ventricular ejection fraction of 30.9% and an ESVi of 57.2 mL/m². Left ventricular ejection fraction and ESVi remained improved during the 12-month follow-up.

Conclusion: This case report demonstrates that VPD implantation in this particular patient was feasible and that it may provide a nonsurgical approach to prevent or reverse left ventricle remodeling. (J Cardiac Fail 2007;13:517–520)

Key Words: Remodeling, ventricular partitioning device.
A 48-year-old man had an anterior MI in 2004 with a subsequent occurrence of LV anteroapical wall motion abnormalities. The patient did not have chest pain but reported dyspnea on heavy exertion, and his condition was evaluated as New York Heart Association class II. In 2005, coronary angiography was performed for occlusion of the proximal segment of the left anterior descending artery with no stenosis on other major epicardial vessels. Echocardiography revealed a dilated LV (62 mm) with anteroapical wall motion abnormalities, no apical thrombus, a calculated ejection fraction (EF) of 26.8% (by Simpson biplane formula), and an end-systolic volume index (ESVi) of 76.8 mL/m². Physical examination and routine laboratory test results were unremarkable. The patient was receiving optimal therapy for heart failure.

The details of VPD implantation have been published. Briefly, implantation was performed through the right femoral artery using a 14F guide catheter. The collapsed VPD was attached to the delivery catheter and advanced retrograde through the guide catheter across the aortic valve and positioned in the LV apex. An LV gram was performed to assess the appropriate positioning. The sizing of the VPD is achieved by measuring the LV diameter 4 cm proximal to the apex, with particular attention to its position relative to the papillary muscles. It is recommended that the VPD diameter exceeds the LV diameter by 30% to 50% (ie, for an LV diameter of 59 mm, as in this patient, a VPD diameter of 85 mm is recommended). Once in place (Fig. 2A), the VPD was expanded by the compliant balloon located proximal to the screw connector (Fig. 2B) and released using a distal screw mechanism (Fig. 2C). The tip of each VPD strut ends in a 2-mm long anchor similar to a “fish hook.” The purpose of these anchors is to engage the tissue, stabilize the VPD, and prevent dislodgment and migration of the VPD after the VPD is detached from the delivery catheter. A control LV gram demonstrated that the VPD was positioned at the apex, with no residual leak between the walls of the LV and the device (Fig. 2D). LV end-diastolic pressure decreased from 18 mm Hg preimplant to 14 mm Hg immediately postimplant. The total procedure time was 75 minutes, and fluoroscopy time was 14 minutes.

The postimplantation course was uneventful, and the patient was discharged after 2 days. Echocardiography on discharge showed the VPD implanted at the apex, with a left ventricular ejection fraction (LVEF) of 30.9% and an ESVI of 57.2 mL/m². The patient was discharged with aspirin, fosinopril, carvedilol, hydrochlorothiazide, and simvastatin, with the addition of low-dose Coumadin (target international normalized ratio of 2.0) for 3 months and clopidogrel for 6 months to prevent VPD thrombosis. After 9 months, computed tomography using 2-mm slices showed a mass consistent with thrombus on the apical portion of the VPD and a thin layer on the ventricular side consistent with endocardial overgrowth of the device (Fig. 3). Figure 4 demonstrates that the LVEF and ESVI remained improved during the 12-month follow-up. The patient’s functional capacity improved and is currently evaluated as New York Heart Association I class.

LV remodeling leads to increased myocardial wall stress, which results in increased myocyte stretch, myocardial oxygen consumption, decreased myocyte shortening, and reduced subendocardial perfusion. The most direct approach to the remodeling itself is a mechanical intervention to decrease the LV volume or constrain the ventricle from further enlargement. Several surgical approaches have been advocated in patients with a dilated LV, including partial left ventriculectomy (Batista procedure), cardiomyoplasty, and surgical ventricular remodeling (Dor procedure). Of these procedures, only surgical ventricular remodeling is frequently used with acceptable short and long-term effects on mortality and morbidity. Still, preoperative mortality is more than 5%, and the overall 5-year survival is less than 70%.

The other approach to mechanical intervention includes the use of cardiac support devices. However, implantation of cardiac support devices requires open surgery. These devices can be divided into groups that acutely alter ventricular shape or geometry (eg, the Myosplint device (Myocor, Maple Grove, Minn) and devices that reverse LV remodeling in a slower fashion through a reduction in wall stress (eg, the CorCap Cardiac Support Device; Acorn Cardiovascular, St. Paul, Minn). The CorCap device, which is a multifilament polyester mesh implant that is placed around both ventricles, has been studied extensively, and sustained decrease in LV volumes and improvement in LVEF have been shown over long-term follow-up.

Preclinical studies in sheep models of MI have indicated that the VPD has beneficial effects on LV function, as measured by a decrease in LV volumes and end-diastolic pressure, increased cardiac output, and improvement in EF. Theoretically, the VPD provides at least 2 clinical advantages over the currently used mechanical interventions. First, the VPD is implanted percutaneously, thus avoiding early mortality and morbidity associated with surgical intervention, and can be more readily used in patients in whom surgical revascularization is not an option. Second, the VPD can be used early after an MI, with or without percutaneous coronary intervention, to prevent extensive LV remodeling.

Therefore, it can be speculated that the combination of standard medical therapy and percutaneous volume reduction may be an important option to consider in the treatment of patients with anteroapical wall motion abnormalities. An international multicenter trial that will examine the

Fig. 1. Detailed description of the VPD. Courtesy of CardioKinetix Inc., Redwood City, California.

Discussion
Fig. 2. Implantation of the VPD showing positioning of the collapsed VPD at the LV apex (A), VPD expansion (B), VPD release (C), and control left ventriculography at end diastole (D) and end systole (E). See text for details.
long-term effects of VPD implantation in 40 patients is currently under way.

In conclusion, this case report demonstrates that VPD implantation in this particular patient was feasible and that it may provide a nonsurgical approach to prevent or reverse LV remodeling.

References


